Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, held at the New York Academy of Medicine, Tuesday evening, April 3d, 1894.

Dr. M. ALLEN STARR, President, in the chair.

A CASE OF AMYOTROPHIC LATERAL SCLEROSIS.

Presented by Dr. CHARLES HENRY BROWN.—The patient was a boy, aged fifteen, who two and one-half years ago began to notice first a loss of proper speech, difficulty in whistling and in moving the tongue. These symptoms were rapidly followed by difficulty in deglution, closing of the eyes, deafness and inability to move the facial muscles. After a few weeks he was unable to use the fingers freely in buttoning and unbuttoning his clothing. At present the patient is extremely emaciated. There is paralysis of the seventh nerve on both sides. Trophic degeneration in the muscles of the face and neck. The condition known as " Taper mouth " is present. The tongue is very much atrophied. The larynx is distorted. He presents all the symptoms of glosso-labio-laryngeal paralysis and nuclear implication of the bulb. There is atrophy of numerous muscles in the upper extremity, and of a few in the lower extremity. There are general fine and coarse fibrillary twitchings all over the body ; exaggeration of superficial and deep reflexes and slight tonic and spastic action in the movement of many muscles.

A CASE OF PROGRESSIVE MUSCULAR DYSTROPHY.

Presented by Dr. ALFRED WIENER.—The patient was a male, aged twenty years. He was in good health up to May, 1893, when he began to suffer considerable pain in the region of the liver and spleen. This was most severe on walking. Soon afterwards he began to experience difficulty in going up-stairs, and he noticed that he was growing very much thinner. His weakness was at first confined to the muscles around the thigh; from here it spread up along the back and involved the muscles of the neck and shoulders. There was no vesical or rectal trouble. There is no history of alcoholism or syphilis. Family history negative.

The patient's present condition is as follows: No mental symptoms. Voice and speech appear to be normal. He is very much emaciated, especially in the neighborhood of the shoulders, back and thighs. He assumes the position of one with a marked lordosis, and walks with a waddling gait. On lying down it is impossible for him to turn over or lift his head from the pillow. His muscles are soft and flabby to the touch. There are no contractures nor fibrillary twitchings; no vasomotor or trophic disturbance. The thoracic and abdominal organs appear to be in perfect condition. There is present no pseudo-hypertrophy in any of the lower muscles, nor does the patient give a history of any having existed. The deep reflexes are all much diminished. The nerves are not painful to pressure, and there is no spinal tenderness. The lordosis is due to the paresis of the muscles of the dorsal and lumbar portions of the back.

Dr. JOSEPH COLLINS said that the case presented by Dr. Brown was very similar to one under his observation at the present time, excepting that his patient was a man thirty-four years old, in whom the symptoms came on rapidly about six months ago. It is very uncommon to see the disease at such an early age as that of Dr. Brown's patient, but he thought that cases showing the occurrence of the disease at even a more tender age have been recorded. The involvement of the fifth nerve that Dr. Brown's patient seemed to present he had never before heard of. The patient presented by Dr. Wiener seemed to be a typical one of muscular dystrophy, and one whose pathognomonic symptoms were in marked contrast to the case of amyotrophic lateral sclerosis shown by Dr. Brown.

The PRESIDENT said that the contrast between the cases shown by Drs. Brown and Wiener was interesting; one presented the typical features of amyotrophic lateral sclerosis, namely, atrophy, fibrillary twitchings, increased reflexes and bulbar invasion; the other showed a pure dystrophy, without twitching and without change of reflexes.

Dr. E. D. FISHER said the cases presented by Drs. Brown and Wiener represent two distinct classes of diseases. The first shows apparently a rare condition in which an inflammation has affected the bulb and later the anterior horns in the cervical region, with involvement of the lateral columns. In other words, we have amyotrophic lateral sclerosis with bulbar symptoms. The usual and not uncommon order of sequence is that of involvement of the cord, with extension later to the medulla. Dr. Fisher said a patient recently under his observation, aged twenty, presented bulbar symptoms of rather acute onset; death resulted suddenly, probably from some involvement of the respiratory centre in the medulla. The autopsy revealed softening in the region of the glosso-pharyngeal and pneumogastric nuclei. That case would probably have presented a similar clinical history, if the patient had lived.

Dr. SACHS said he agreed with the diagnosis of amyotrophic lateral sclerosis in Dr. Brown's case. Dr. Wiener's case was undoubtedly one of progressive muscular dystrophy.

Dr. WILLIAM M. LESZYNSKY called attention to the fact that in Dr. Wiener's case the upper part of the trapezius muscle was atrophied, while the lower part escaped. This partial atrophy, he said, usually occurs in progressive muscular atrophy of spinal origin.

Dr. SACHS said that the partial atrophy of muscles may also occur in spinal dystrophies.

The PRESIDENT said that in three cases of amyotrophic lateral sclerosis coming under his observation, the bulbar symptoms were very prominent.

Dr. SACHS expressed the opinion that the bulbar symptoms in amyotrophic lateral sclerosis were due to a natural extension of the disease upwards. He inquired whether there was involvement of the eye in these cases.

Dr. BROWN said there may be slight ophthalmoplegia.

Dr. L. C. GRAY said he has seen the eye involved in one case.

Dr. BROWN, in closing the discussion, said he presented his case as a typical one of amyotrophic lateral sclerosis, but rare in the fact that it had commenced in the bulb and extended downwards. He thought this type of nuclear trophic and motor disorder was somewhat acute in its manifestations, and rapidly ran its course. He considered that his case was in *statu quo*, and would progress no further so far as degeneration of muscles was concerned. The boy presented a typical picture of a glosso-labio-laryngeal paralysis, which is an extremely rare condition in childhood. The fibrillary twichings and slight spastic actions and increase of the reflexes undoubtedly contribute to the picture of a lateral sclerosis.

The PRESIDENT exhibited a diagram showing the areas of anæsthesia in the arms as the result of lesions involving different segments of the cervical and dorsal portions of the cord. This diagram, he said, was the result of a careful study of a large number of cases—either coming under his own observation, or reported by others—in which the spinal cord was involved. The result seems to show that for each segment of the cord there is an area of the skin which is anæsthetic when that segment becomes involved.

A CRITICAL REVIEW OF THE VARIOUS THEORIES OF URÆMIA, BASED UPON ORIGINAL EXPERIMENTAL OBSERVATIONS.

Dr. C. A. HERTER read a paper on this subject. He began by stating that few subjects of a medical nature have received more attention at the hand of clinicians and investigators, than that of uræmia. Yet at the present time there is an uncertainty as to the nature of the uræmic state that is discouraging, both to the student of pathology and the practitioner who seeks to understand the conditions he is called upon to treat.

Dr. Herter first gave a brief historical sketch of the growth of opinion regarding the nature of uræmia, and then reviewed in detail the various theories that have been advanced. The first one taken up was that known as the mechanical theory of uræmia, which has its chief basis in the clinical and post-mortem studies of Traube, who was impressed with the facts that in many cases of Bright's, the blood is impoverished in its corpuscular and proteid elements, the left ventricle is hypertrophied, and the arterial tension is greatly increased. The hydræmia combined with high arterial pressure was supposed to account for the cerebral œdema found at autopsy. The uræmic symptoms, Traube referred not to cerebral œdema, but to the anæmia of the brain, resulting from the pressure exerted by this œdema. There are objections to this theory. In the first place, œdema of the brain is present in only a small proportion of cases where there have been unequivocal symptoms of uræmia during life. There have been many cases of kidney disease with no hydræmia, no cardiac hypertrophy and no increase of arterial tension; yet even when these latter conditions are present, there is usually no œdema of the brain. In the second place, œdema of the brain is found in conditions where Bright's is absent, and there are no symptoms of uræmia. Then again, drugs which produce convulsions may produce cerebral œdema, and it is likely that the œdema of the brain seen in uræmics is often the consequence rather than the cause of the convulsions. A further objection to the mechanical theory is that lowering of the arterial tension, where it is high, does not necessarily relieve the uræmic symptoms. The dyspnœa of Bright's is often thus relieved, but usually not the convulsions. Bleeding frequently stops the latter, but it checks them often also where there is no excess of tension. The effects of bleeding cannot be used as an argument in favor of Traube's theory, because it may be claimed that the bleeding relieves the circulation of poisonous substances.

The carbonate of ammonium theory. This was advanced by Frerichs, and is based upon the following propositions:

1. It is a well-known property of urea to be readily transformed under favorable circumstances into ammonium carbonate.

2. Carbonate of ammonium can always be detected by chemical means in the blood of uræmic patients.

3. The injection of ammonium carbonate into the blood of animals gives rise to the symptomic group of uræmia.

The fate of Frerichs' theory hangs on his second proposition, namely, that the blood of uræmics always contains ammonium carbonate. From numerous experiments made by Gobee, Oppler, Zalewsky, and others, including the author, the following conclusions may be drawn: 1. That no ammonium carbonate, or only a small amount is found in the blood of uræmic persons. 2. That amounts of ammonium carbonate far smaller than the quantity required to kill are readily detected. 3. That urea injected into the blood of dogs is not converted into ammonium carbonate. We may, therefore, state that Frerichs' second proposition is without a substantial foundation. It has been stated that the ammoniacal breath occasionally met with in uræmic patients is evidence of ammonia in the blood. It is more liable to be found in uræmics with gastro-intestinal symptoms, and the ammonia probably comes not from the blood, but from the gastro-intestinal tract.

The theory of Trietz differs from that of Frerichs mainly, in that it supposes the conversion of urea into ammonium carbonate to take place in the intestine instead of the blood. He holds that it is by the entrance of ammonium carbonate into the blood that the uræmic state arises, and objections already made to the proposition of Frerichs, that uræmic blood contains ammonium carbonate, apply here with equal force.

The potassium theory of Felz and Ritter is based upon the idea that the potassium of the blood-serum, which is normally present in very small amount, might accumulate under pathological conditions and cause death, the potassium salt being rapidly fatal in animals when injected into the veins in even inconsiderable quantity. The experimental and chemical evidence of various observers is strongly opposed to this theory, and it may be unhesitatingly abandoned.

The theory of Brown-Sequard. According to this writer, the kidney elaborates an internal secretion which is essential to health, and the suppression of which is responsible in a large degree for the phenomena of uræmia, while the accumulation of toxic substances in the blood is thought to have little or no influence in causing uræmic symptoms. The following are the chief facts upon which this hypothesis rests: 1. It is claimed that the injection of kidney extract into the circulation of a nephrectomized dog causes the temporary disappearance of uræmic symptoms. 2. It is held that the well-authenticated cases of survival of patients with anuria for a week or more without any signs of uræmia, especially in cases of mechanical obstruction of the ureters, is evidence that it is owing to the internal secretion of the kidney that this inhibition of uræmic symptoms takes place. All the facts advanced in support of the first proposition, Dr. Herter said, are equivocal, and those advanced to support the internal secretion theory are equally weak.

The theory of extractives and toxines. The extractive theory of uræmia refers the toxæmic symptoms to the accumulation in the blood of the extractives usually found in urine; among these may be mentioned xanthine, uric acid, creatinine, etc. In order to show that a substance plays a part in the production of uræmic symptoms, it is necessary to prove that this substance is present in the blood in uræmia, or is present in excessive amount, and that the substance is toxic in the higher mammals. The evidence is not fully satisfactory regarding either of these points. The observations that have been made hardly establish the fact that an accumulation of the extractives is a feature of all, or many cases of uræmia. The evidence is even less strong in regard to particular members of the extractive group.

Regarding the view which attributes the uræmic phenomena to the action of toxines, the poisonous basic products of bacterial activity, there is little to be said, for the evidence on which it is built is scanty and conflicting. None of the toxic ptomaines that have been suspected in this connection have been found in the blood of uræmics.

The urea theory of uræmia. The evidence relating to urea as a factor in uræmia may be grouped in answer to the following queries: 1. Does urea occur in excess in the blood of uræmic patients? 2. Is urea toxic, and if so, to what extent does it explain the symptoms of uræmia? As regards the first question, we have a sufficiently large accumulation of observations made by competent investigators to enable us to reach a definite decision. In the blood of Bright's disease, the quantity of urea is largely increased beyond that found in the normal blood, and its presence is readily detected even by imperfect chemical methods. When we come to the second question, we find that the effects which favor and those which oppose the idea that urea is toxic seem to be about evenly balanced. Dr. Herter then reviewed the observations that have been made bearing on this question, and detailed a number of experiments made by himself, which prove that pure urea injected into the blood of the dog and monkey in large amount is in the highest degree toxic. The autopsies in these cases disclosed marked congestion of the gastro-intestinal tract. To what extent and under what conditions urea is a factor in uræmia, the author said it was not possible for him to state at the present time. Facts at our command strongly favor the view that the gastro-intestinal symptoms of uræmia are due to urea. There is no doubt that we at present group together under the term "uræmia" conditions which are totally distinct as regards pathology. The evidence is very strong, though not quite conclusive, that one group of symptoms depends largely on the accumulation of urea, and, perhaps, extractives in the blood, while another set of symptoms bears the stamp of an infective process, operating in the presence of renal insufficiency. Observations may show that even further subdivisions of cases are necessitated by pathological considerations.

It is upon the following facts that the author based the view that urea plays an important part in causing the gastro-intestinal symptoms of uræmia:

1. The presence of urea in excess in the blood in such cases.
2. The property which pure urea possesses of causing such symptoms when injected in the circulation.
3. The occurrence in Bright's of congestion of the gastro-intestinal tract, and the occurrence of a similar congestion in animals, which is positively due to the injection of urea into the blood.
4. The fact that urea is found in the dejecta of patients with gastro-intestinal uræmia.
5. The absence in many of these cases of any elevation of temperature, or other evidence of an acute toxæmia.
6. The fact that the urine in these cases, even at the time of the crisis, is no more toxic than normal urine, which is in sharp contrast to the very toxic urines found in acute febrile uræmias of cerebral type.

Dr. W. H. THOMSON said the subject of uræmia has always been and still remains a very mixed one in his mind. Dr. Herter's paper is certainly a very valuable contribution to the subject. The cases of uræmia to which Dr. Herter principally confined himself—those in which the characteristic symptoms are vomiting and purging—form comparatively a small minority of those that come under our observation. In many cases there are no gastro-intestinal symptoms whatever. Theoretically, Dr. Thomson said, he still felt strongly inclined to the view that toxines play a very important part in the production of the purely nervous symptoms of uræmia. Regarding the experiments performed by Dr. Herter, in order to prove the toxic effects of urea, Dr. Thomson inquired whether the injection into the blood of sodium chloride, or other similar substances, might not give rise to derangements of the system to some degree resembling those produced by the repeated and rapid introduction of increasing quantities of pure urea.

DR. WILLIAM H. FLINT said he was fully in accord with the statements made by Dr. Herter in so far as they went to prove the etiology of certain forms of uræmia.

Dr. J. S. ELY referred to the intestinal lesions that he had observed in performing autopsies on persons who died of nephritis. It is often difficult to state positively whether the conjestion was present before death or produced by putrefactive changes. Unless the autopsy is made very soon after death it would be hazardous to argue from the redder appearance of the mucous membrane that the congestion was the result of the disease.

Dr. B. SACHS expressed the hope that the critics of the future would deal more leniently with Dr. Herter's theory than he had dealt with the theories reviewed in his paper, and they would expend the same amount of thought and labor to prove their assertions. He did not think that anyone should attempt to establish a single theory to explain such a complex of symptoms as we have in uræmia. Very much the same symptoms occur under other conditions, and we are perfectly willing to recognize that different morbid processes give rise to them.

Dr. HERTER then closed the discussion. He stated that in his paper he did not advocate any one theory to explain all the phenomena of uræmia. What he did advocate was that there is a certain class of symptoms, a limited class, namely, the gastro-intestinal symptoms, which are met with in certain cases of uræmia, and which are due, in all probability, to urea. The proof of this is not absolutely satisfactory, but he did not see how the proofs adduced by his experiments could be interpreted in any other light. In reply to Dr. Thomson's question, Dr. Herter said that large quantities of sodium chloride can be injected into the blood without producing results comparable to those produced by the introduction of urea. As regards the cerebral symptoms of uræmia, he does not know what they are due to, and in his paper he made no attempt to throw any light on those cases. The autopsies on the animals which were killed by the introduction of urea were performed immediately after death, and the intestinal congestion was very marked.

ELECTION OF OFFICERS.

The following officers were elected for the ensuing year: President, Dr. E. D. Fisher; Vice-President, Dr. C. A. Herter; 2d Vice-President, Dr. Wm. M. Leszynsky; Recording Secretary, Dr. Frederick Peterson; Corresponding Secretary, Dr. Mary Putnam-Jacobi; Treasurer, Dr. G. M. Hammond.

PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, March 26th, 1894.

Vice-president, Dr. JAMES HENDRIE LLOYD, in the chair.

Dr. M. ALLEN STARR, of New York, read a paper on

LOCAL ANÆSTHESIA AND OTHER SYMPTOMS PRODUCED BY LESIONS OF THE CERVICAL CORD.

ABSTRACT.

AFTER alluding to the relations between the various segments of the cervical cord and the spines and bodies of the cervical vertebræ, and showing that the relation is a variable one, the author proceeded to cite a number of cases of limited lesion of the cervical cord, traumatic, hemorrhagic, inflammatory and due to tumor, in which the local symptoms had been exactly studied, and in which the anæsthesia present had been carefully laid down on diagrams.

He then drew the following conclusions:

CONCLUSIONS.

First. As to the distribution of anæsthesia in lesions of the cervical enlargement of the spinal cord. By combining the diagrams already given with numerous other diagrams published by Thorburn and others, and comparing the results with descriptions of numerous cases